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☐ 1: Arch Toxicol Suppl 1996;18:127-39

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Cytokines in human lung fibrosis.

Martinet Y, Menard O, Vaillant P, Vignaud JM, Martinet N.

INSERM U 14, Nancy-Vandoeuvre.

Fibrosis is a pathological process characterized by the replacement of normal tissue by mesenchymal cells and the extracellular matrix produced by these cells. The sequence of events leading to fibrosis of an organ involves the subsequent processes of injury with inflammation and disruption of the normal tissue architecture, followed by tissue repair with accumulation of mesenchymal cells in the area of derangement. The same sequence of events occurs in wound healing with normal granulation tissue and scar formation, but, while normal scar formation is very localized and transient, in contrast, in fibrosis, the repair process is exaggerated and usually widespread and can be chronic. Inflammatory cells (mainly mononuclear phagocytes), platelets, endothelial cells, and type II pneumocytes play a direct and indirect role in tissue injury and repair. The evaluation of three human fibrotic lung diseases, two diffuse [idiopathic pulmonary fibrosis (IPF), and the adult respiratory distress syndrome (ARDS)], and one focal (tumor stroma in lung cancer), has shown that several cytokines participate to the local injury and inflammatory reaction [interleukin-1 (IL-1), interleukin-8 (IL-8), monocyte chemotactic protein-1 (MCP-1), tumor necrosis factor-alpha (TNF-alpha)], while other cytokines are involved in tissue repair and fibrosis [platelet-derived growth factor (PDGF), insulin-like growth factor-1 (IGF-1), transforming growth factor-beta (TGF-beta), and basic-fibroblast growth factor (b-FGF)]. A better understanding of the cytokines and cytokine networks involved in lung fibrosis leads to the possibility of new therapeutic approaches.

Publication Types:

- Review
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PMID: 8678788 [PubMed - indexed for MEDLINE]

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